Both spontaneous and positional nystagmus are important neurologic signs, but they can be more easily identified at the bedside than in the laboratory provided the ability of the patient to use visual fixation to suppress nystagmus is eliminated. This can be accomplished easily with Frenzel glasses (internally illuminated goggles containing high-magnification spectacle lenses). Frenzel glasses allow the examiner to see the eyes of the patient but prevent the patient from fixing on objects. Thus, by eliminating fixation, any inherent spontaneous vestibular nystagmus becomes manifest and can be detected by the examiner. It must be remembered that small amounts of spontaneous and especially positional nystagmus can be recorded with an ENG in many normal persons.

The response to caloric stimulation is a measure of dynamic vestibular function. It is probably the most useful information that can be gleaned from an ENG, with the following caveats:

- Caloric testing assesses the function of the lateral semicircular canals only—not of the vertical canals or the otolith organs.
- The range of values of the response to caloric irrigations in normal persons is wide both in amplitude and symmetry. Consequently, the caloric test is relatively insensitive to partial labyrinthine lesions.
- The caloric test is subject to technical problems, such as the presence of cerumen, that may cause difficulty in simultaneously delivering an equal quantity of water at the same rate and temperature to each ear. If such sources of artifact are not kept in mind, decreased vestibular function in one or both ears may be mistakenly inferred. Caloric testing must be carried out by experienced personnel paying rigorous attention to careful technique.
- Caloric irrigations mimic a head rotation that is composed only of low frequencies. Patients with absent caloric responses due to bilateral peripheral vestibular lesions may still show a normal response to a rotational stimulus that contains high-frequency components.

The presence of a rotational, but not a caloric, response has important functional and prognostic implications. Thus, while rotatory chair testing is rarely helpful in lateralizing vestibular lesions, it sometimes provides important functional information that complements or confirms the results of caloric testing.

The caloric test is most useful when it is done in a high-quality laboratory and when it unambiguously reveals a unilateral or bilateral vestibular loss. Normal or mildly abnormal results on caloric testing do not allow the physician to assume that the patient does or does not have a vestibular lesion.

As in most areas of medicine, a careful history and physical examination, couched in a solid understanding of pathophysiology, is the way to diagnose accurately a vestibular disorder. The physician must specifically ask about oscillopsia—visual blurring or jumping—during head motion, which points to an abnormality in reflexes mediated by the semicircular canals. The physician must specifically ask about sensations of translation or tilt, which point to an abnormality in reflexes mediated by the otoliths. The physician must specifically ask about vertigo induced by Valsalva's maneuvers, which points to a perilymph fistula or to a craniocervical junction anomaly. Both are amenable to surgical treatment.

Perhaps the most common cause of vertigo is benign paroxysmal positional vertigo, which is diagnosed from a characteristic history and physical findings.[1] No laboratory tests are necessary to make this diagnosis, yet most of such patients undergo unnecessary magnetic resonance imaging and computed tomographic scans and electronystagmography.

Even such a basic part of the physical examination as the testing of hearing is often neglected in patients with dizziness. A unilateral hearing loss, with or without vestibular symptoms, always requires further evaluation for an acoustic nerve tumor. In fact, the most useful laboratory test in evaluating patients with vestibular dysfunction is probably the audiogram, just as vision acuity must always be measured in patients whose symptoms point to optic nerve dysfunction.

Finally, I would like to emphasize an old, simple bedside test for vestibular dysfunction that we have recently resurrected and found to be extremely useful—the induction of nystagmus after 20 seconds of vigorous head shaking.[2] When used with Frenzel lenses, this test is probably as sensitive and as reliable as the caloric test in identifying a unilateral peripheral vestibular lesion.

To summarize, vestibular function tests have a place in the evaluation of patients with dizziness and disequilibrium, but for most patients the correct diagnosis is reached by a thoughtful, physiologically oriented history and examination. With the vast number of people who are bothered by dizziness, imbalance, or both, perhaps our medical school curriculum committees should recommend more attention to the teaching of vestibular physiology and pathophysiology. How else will physicians not only understand new developments in the laboratory evaluation of vestibular function but also correctly interpret their patients' symptoms and elicit the appropriate diagnostic signs?

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Clinical and Laboratory Assessment of Patients With Possible Hemorrhagic Disorders

ALTHOUGH IT HAS ALWAYS BEEN OF CRITICAL IMPORTANCE to determine whether a patient has a defect in primary or secondary hemostasis, this clinical problem is even more urgent at the present time, because therapy for the arrest of hemorrhage with blood products should be avoided whenever possible. Disorders of primary hemostasis are mainly characterized by bleeding into superficial areas of the skin and mucous membranes and the cause is usually attributable to quantitative or qualitative platelet defects. Disorders of secondary hemostasis involve muscle tissues with dissection into fascial planes, large ecchymotic lesions in the skin, and hemarthroses.[1,2]

Patients always require screening evaluations before an operation or on being seen by a physician after an episode of hemorrhage of either a spontaneous nature or following trauma or a medical procedure. A detailed personal and ge-

netic history, superimposed on the findings of a complete physical examination, will frequently corroborate subsequent laboratory screening procedures. These should be carried out before any further medical treatment is pursued or before the patient is told that he or she may resume routine activities. Skin bruising that occurs spontaneously or in relation to minor trauma is significant because it suggests that a hemorrhagic abnormality exists. In platelet disorders, bleeding is immediate but temporary and blood loss is usually minimal. This is in contrast to disorders of coagulation, such as factor VIII or IX deficiency where hemorrhage is delayed, somewhat prolonged, and in the moderate to severe category. About 85% of congenital coagulation disorders are due to deficiencies in factor VIII (hemophilia A). About 10% are attributable to factor IX deficiency (hemophilia B), and the other coagulation defects make up the additional 5%.[1,3]

In general, if an asymptomatic patient is being evaluated before a major operation and the history is negative, the screening laboratory tests need not be elaborate.[3] Under these conditions, a platelet count, an activated partial thromboplastin time (PTT), and a prothrombin time are recommended. On the other hand, if a hemorrhagic diathesis is suspected from the history and physical examination, laboratory procedures that evaluate platelet integrity and the vascular and coagulation components of the hemostatic mechanism are necessary.

- Platelet enumeration and morphology, as well as information on the other hematologic cells from stained blood smears, are important. Erythrocyte abnormalities, such as schistocytosis, are seen in microangiopathic hemolytic anemias and can be associated with disseminated intravascular coagulation (DIC). Large platelets (megathrombocytes) suggest increased platelet turnover in response to hemorrhage, platelet destruction, or immune thrombocytopenia. The most common cause of hemorrhage in the general population is thrombocytopenia. Therefore, this diagnosis should be entertained and ruled out initially. All medications ingested recently should be evaluated as possible causes of thrombocytopenia.
- The bleeding time, appropriately determined, is almost infallible for the diagnosis of a defect in primary hemostasis. If a patient has a normal bleeding time, a platelet disorder is unlikely. A prolonged bleeding time in a patient with a normal platelet count signals a qualitative platelet abnormality and also mandates the diagnostic procedures for von Willebrand's disease. This can be pursued by measuring the components of the factor VIII complex—that is, factor VIII antigen, factor VIII coagulant activity, and factor VIII ristocetin cofactor activity as manifested by agglutination of the patient's platelet-rich plasma on adding the antibiotic, ristocetin. About 43% of patients who have a prolonged bleeding time and a normal platelet count will have von Willebrand's disease. About 27% fall into the category of vascular purpuras (platelets and coagulation normal), 16% have thrombasthenia, 7% have a "thrombocytopathy" with platelets that appear grossly abnormal, and 7% have thrombocytopathy with normal-appearing platelets. The latter two groups can also be classified as having "storage pool diseases." In these patients, a surreptitious ingestion of aspirin-containing drugs must always be ruled out, if possible.
- The prothrombin time is one of the most useful tests in clinical medicine and one that will help diagnose a coagulation defect. This test provides an overall assessment of the extrinsic pathway of coagulation. It is also used to monitor patients on anticoagulant therapy, since factor VII is the first protein to be depleted by oral anticoagulants. The prothrombin time is prolonged in deficiencies of factors II, V, VII, X, and fibrinogen. Circulating anticoagulants may also prolong the prothrombin time, and the test is also valuable for monitoring liver disease.
- The activated partial thromboplastin time (PTT) is the most important screening test for evaluating the intrinsic coagulation system. The "contact" components of the intrinsic system (factor XII, high-molecular-weight kininogen and prekallikrein) are activated by the particulate components of the PTT reagent. I should add that patients whose PTT is prolonged by the latter deficiencies are not symptomatic, and the laboratory abnormality is of no clinical significance. The phospholipid component of the PTT reagent is critical for activating factors V, VIII, IX, and XI. Even a slightly prolonged PTT must be explained before the patient is allowed to undergo any surgical procedures. The test is sensitive to factor deficiency conditions of less than 30% activity (normal range, 50% to 150%).[2]
- The thrombin time evaluates plasma coagulation on adding thrombin. Abnormalities of the fibrinogen molecule—both quantitative and qualitative—and also the presence of inhibitors will influence the thrombin time. Circulating anticoagulants, fibrinogen or fibrin degradation products, or paraproteins also lengthen the thrombin time. When the thrombin time is abnormal, confirmatory tests for identifying fibrinogen or fibrin split products should be carried out. If available, tests for fibrin monomer-fibrinogen complexes in plasma are also valuable, especially if a clinical suspicion of DIC requires confirmation.

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